Arsenic Upsets Heartbeat

Possible Early Warning for Cardiovascular Risk

Acute arsenic exposure can cause severe heartbeat abnormalities, and chronic exposure has been linked to coronary disease and cancer. Now researchers from Inner Mongolia, China, and the United States have begun to analyze the effects of chronic exposure on the electrical signals that regulate heartbeat [*EHP* **115:690–694; Mumford et al.**]. They found a correlation between exposure via drinking well water and signal changes associated with arrhythmia and death.

The heart of the exposure. Many residents of Ba Men, Inner Mongolia, use artesian well water containing high concentrations of arsenic.

Tens of millions of people worldwide drink groundwater contaminated with naturally occurring arsenic. Through metabolism, the inorganic arsenic found in drinking water is converted to more toxic methylated compounds.

The research team focused on the QTc interval, a specific portion of the cardiac signal that corresponds to the active pumping (systole) phase of the heartbeat. QTc intervals of 0.45 second or longer are associated with cardiac risk.

The team used electrocardiography to measure the QTc interval of 168 men and 145 women from four villages in Ba Men, an area of Inner Mongolia where the drilling of artesian wells in 1980 exposed residents to arsenic. Arsenic exposure was determined through analysis of toenail samples from participants and water samples from their wells. Arsenic exposure was categorized as low (21 μg/L or less), medium (100–300 μg/L), or high (430–609 μg/L).

As exposure to arsenic increased, so did the occurrence of prolonged QTc interval, which was seen in 3.9% of the low exposure group, 11.1% of the medium exposure group, and 20.6% of the high exposure group. Women—who typically have a longer QTc interval than men—were more susceptible to this effect than men. Age, smoking, and pesticide exposure did not affect the association.

The authors suggest that arsenic may affect QTc interval by altering the flow of potassium ions that are involved in cardiac signaling. They write that measurement of QTc interval may be useful in the early detection of cardiovascular risk among individuals exposed to arsenic, as well as in the identification of populations where such risk is high. The team is currently conducting a large follow-up study in the same population. Meanwhile, the Chinese government is helping to install water systems in the Ba Men area that will decrease arsenic exposure. **–Kris Freeman**

Short-Term Particulate Threat

Pollution Standard May Not Protect Health

Many studies have shown that particulate matter (PM) poses health risks, yet the attributes of PM that cause these effects remain uncertain. To address some of those critical nuances, especially the short-term effects of specific emissions, researchers used a refined approach, including new application of a pollutant distribution model, to assess links between deaths and two PM components, black carbon and sulfate particles [*EHP* **115:751–755; Maynard et al.**]. They found that as the air concentration of either component increased, there were more deaths the following day. These results occurred even at concentrations below current U.S. standards for fine particulates.

Boston, Massachusetts

Sulfate exposure in the northeastern United States comes in large part from coal-fired power plants. Black carbon is a surrogate for vehicle-related pollution that varies significantly over short distances. The researchers used data from a central monitor at the Harvard School of Public Health to determine concentrations of sulfates and assumed there were homogenous concentrations throughout the study area, a premise other studies have validated. To estimate concentrations of black carbon, they used a model that began its calculations with daily data from another monitor at the school. The model then estimated black carbon concentrations at more than 80 representative sites in the Boston area, incorporating variables such as weather, season, day of week, traffic volume, proximity to major roadways, population density, and percent urbanization. The researchers also accounted for gender, education, income, and residence location for each death.

In evaluating 107,925 deaths that occurred at Boston-area residences from 1995 through 2002, the researchers found that each interquartile increase in black carbon concentration on the day before death was linked with a 2.3% rise in deaths from any cause and a 4.4% increase in stroke deaths. A similar, though smaller, relationship existed for sulfate particles, with each interquartile increase the day before death linked with a 1.1% increase in death from any cause. The researchers also found that for both black carbon and sulfates, there were increases of similar magnitude for deaths from cardiovascular disease, respiratory diseases, and diabetes.

The authors acknowledge that the black carbon estimation model still needs refinement, that the study was limited by its focus on just one city, and that there were relatively limited data for sulfates and some causes of death. Nonetheless, this work confirms past research implicating sulfates and black carbon in the PM–mortality association. As a result, the authors say their findings reinforce concerns that current and proposed fine particulate standards do not adequately protect public health. **–Bob Weinhold**

Top to bottom: Judy Mumford; Dave Rabbin/iStockphoto

Toxic Neighbors?

Fetal Death Risk Near Hazardous Waste Sites

The health effects associated with residential proximity to hazardous waste sites are uncertain, and findings on potential links between prenatal exposure to environmental toxicants and outcomes such as miscarriage are mixed. A recent exploratory study in Washington State finds no evidence for an overall association between hazardous waste sites and occurrence of fetal death, but pesticide-containing sites may be an exception [***EHP*** **115:776–780; Mueller et al.**].

Using state health department records, researchers examined the occurrence of fetal death, defined as pregnancy loss after 20 weeks' gestation, against the straight-line distance between a mother's home and the nearest hazardous waste site. The team used ten live births for each fetal death as controls and considered several factors that could affect pregnancy outcome, such as maternal smoking, alcohol consumption, age, medical conditions, and socioeconomic status. Between 1987 and 2001, the state recorded 7,054 fetal deaths; the team located maternal residences for 5,302 cases and 61,455 controls.

Hazardous waste sites were characterized according to type of contaminant (solvents, metals, pesticides, or radioactive substances) and type of contaminated medium (air, water, or soil). Sites were also ranked as "high priority" or "low priority" depending on their potential hazard to public or environmental health.

Maternal characteristics more common among women who experienced a fetal death included being unmarried and older than 35, having less than a high school education, drinking alcohol during pregnancy, and receiving government-funded medical assistance. Mothers who experienced fetal death were also more often of nonwhite race/ethnicity and less likely to have had a previous pregnancy or birth. In general, no association was seen between hazardous waste site proximity and fetal death. However, subanalysis by contaminant type showed a small but significant increase in fetal deaths within five miles of pesticide-contaminated sites, with a slightly increased risk with each mile nearer such waste sites. Subanalysis by priority type revealed a slight but nonsignificant increase in fetal death for mothers within two miles of a high-priority site.

The authors describe several study limitations. No actual toxicant exposure measurements were available, potential occupational exposures and their duration were unknown, and fetal deaths and pertinent maternal health information may have been underreported. The findings do not negate the need for waste site remediation, however, and in light of other research showing health risks linked to prenatal pesticide exposure, the authors recommend that more attention be paid to pesticide-contaminated sites. **–Julia R. Barrett**

Cottonwood Clues in Fallon

Tree Rings Reflect Tungsten, Cobalt Exposure

The cause of a childhood leukemia cluster in Fallon, Nevada (population 8,000) has mystified investigators since it was first discovered in 2000. Sixteen children have been diagnosed with acute lymphocytic leukemia and one with acute myelocytic leukemia. Because known risk factors such as ionizing radiation and prenatal exposure to volatile organic compounds do not explain most acute lymphocytic leukemia cases, researchers suspect other environmental exposures in Fallon. Now a tree ring study reveals elevated environmental tungsten and cobalt levels in Fallon compared to other towns in the area in the years just prior to the onset of the cluster [***EHP*** **115:715–719; Sheppard et al.**].

Among Fallon's potential sources of contamination are a tungsten carbide production facility, melon and alfalfa fields, and a naval air base jet fuel pipeline. The study team took core samples representing the years 1989 through 2002 from cottonwood trees around the Fallon processing plant and analyzed them for tungsten, cobalt (also used in tungsten carbide processing), and a range of other metals. For comparison, they also sampled trees in three nearby towns. In addition, the team tested trees in Sweet Home, Oregon, which also has a known local source of airborne tungsten, to test the dendrochemical technique independently.

Branching out. The cottonwoods around Fallon may add a new chapter to the story of the town's leukemia cluster.

Before 1992, median tungsten levels in Fallon tree rings differed little from those in the comparison Nevada towns, but rose thereafter to levels significantly higher than those in the other towns. Median cobalt levels in Fallon were higher than in surrounding towns but remained constant over the study period. Other trace metals did not increase consistently over time.

It is unknown whether tungsten causes cancer. The National Toxicology Program is currently assessing its disposition in rodents, with carcinogenicity studies planned. Cobalt has been associated with lung cancer, thyroid disorders, and lung disease, according to the CDC's 2005 *Third National Report on Human Exposure to Environmental Chemicals*. The International Agency for Research on Cancer has classified the combination of tungsten carbide and cobalt as a probable human carcinogen.

By itself the tree-ring study does not establish a causal link between these elements and leukemia, but based on the temporal change in tungsten and the high level of cobalt found in the trees, further biomedical research is advisable. **–Valerie J. Brown**

Top to bottom: Jeff Banke/Shutterstock; Paul R. Sheppard